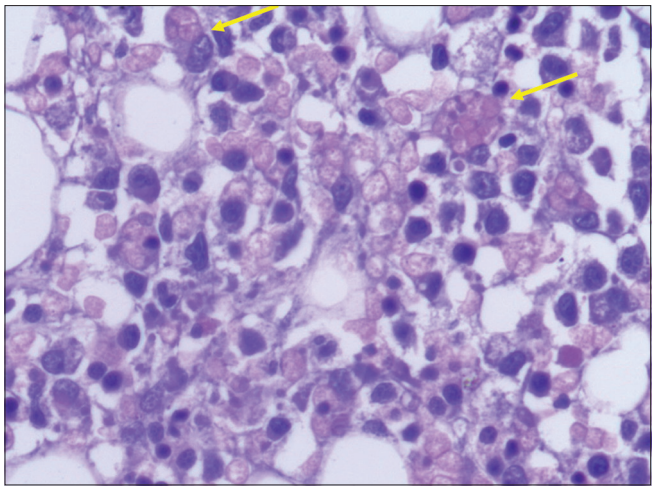

Figure 3: Bone marrow biopsy smear demonstrating hemophagocytosis (hemophagocytes marked with arrows, H and E, ×400)

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

There are no conflicts of interest.

Ting Su, Wenjie Xiong[1], Yang Xu, Yan Lu

Departments of Dermatology and [1]Gastroenterology, The First Affiliated Hospital of Nanjing Medical University, Nanjing, China

Corresponding author:
Prof. Yang Xu,
Department of Dermatology, The First Affiliated Hospital of Nanjing Medical University, Nanjing, China. cardioos@163.com

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Erlotinib-induced reactive perforating collagenosis in a case of lung adenocarcinoma

Sir,

Acquired reactive perforating collagenosis, an uncommon dermatosis characterized by transepidermal elimination of degenerated collagen, generally affects patients with diabetes mellitus, chronic renal insufficiency, Hodgkin's lymphoma, acute leukemia, infestations like scabies, etc. Herein, we report a rare case induced by oral erlotinib in a woman with lung adenocarcinoma.

A 53-year-old female, previously diagnosed with lung adenocarcinoma (Stage III b) which was surgically resected two years back and later initiated on treatment with oral erlotinib 150 mg daily, presented to the dermatology clinic with skin eruptions. She had developed pruritic reddish papules over her face, neck and trunk one week after starting erlotinib and the lesions gradually progressed. Pruritic lesions were noted on the buttocks, perineum, trunk and extremities too, in the past two months. Physical examination revealed scattered or densely distributed reddish follicular papules and papulopustules involving the face, neck and upper trunk [Figure 1a]. Multiple red-colored, umbilicated, dome-shaped papules of size five to ten millimeters were noted on the trunk and extremities, a few of them partly coalescing to form plaques [Figures 1b and c]. Some lesions also exhibited a linear configuration, indicating a Koebner phenomenon. Histopathology from the umbilicated papule showed a cup-shaped depression of the epidermis with a keratin plug showing parakeratosis, inflammatory debris and degenerated collagen fibers and perivascular infiltrate of inflammatory cells below the

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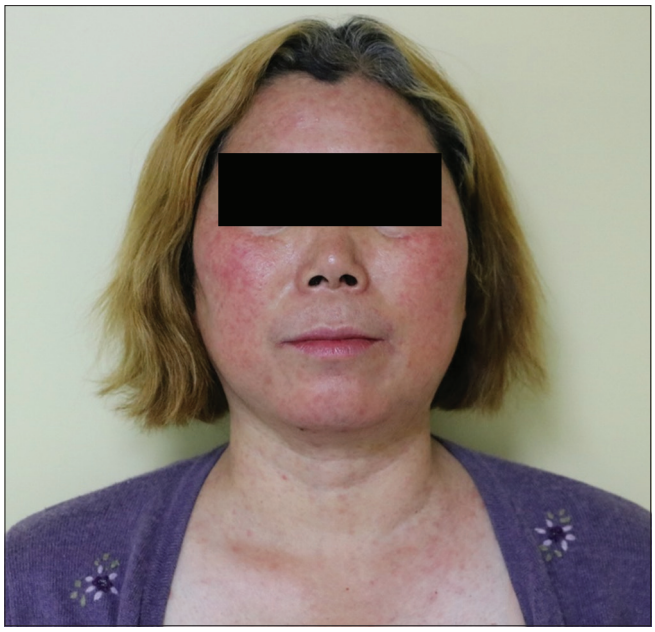

Figure 1a: Reddish follicular papules, papulopustules on her face, neck and upper trunk

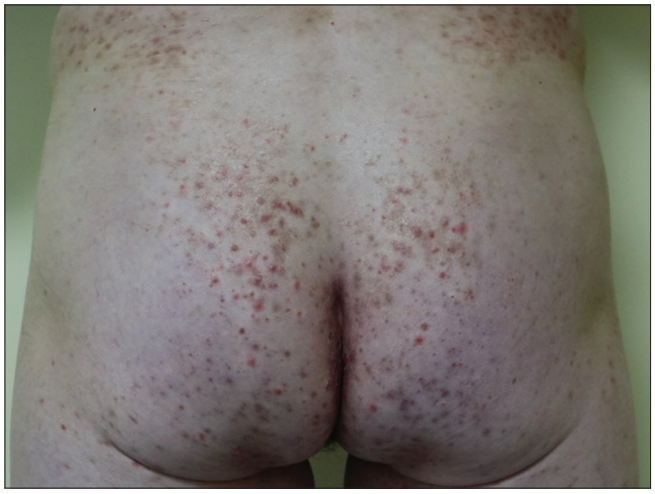

Figure 1b: Multiple red-colored, dome-shaped papules on her trunk and extremities

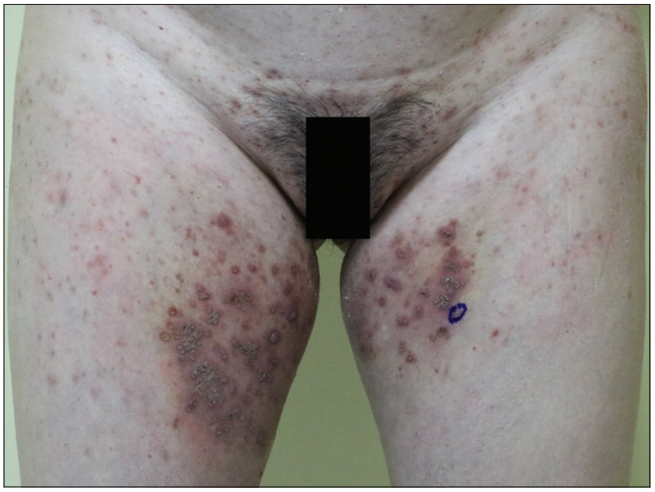

Figure 1c: Umbilicated papules with keratin plugs on her thighs. Koebner phenomenon can be seen

depression [Figure 2a]. The elimination of degenerated collagens fibers through the epidermis could be seen [Figures 2b and c].

A diagnosis of acquired reactive perforating collagenosis with acneiform eruptions was made. She was treated with oral isotretinoin 10 mg twice daily, topical mometasone furoate cream 0.1% and tretinoin cream 0.1% once daily on her trunk and extremities. Most of the lesions regressed completely within two months [Figure 3]. One month later, erlotinib was discontinued following recovery from lung cancer. Isotretinoin and topical drugs were also stopped following that. No relapse was recorded during the 10-month follow-up period after treatment and a long-time follow-up is still being maintained.

Acquired reactive perforating collagenosis was originally described in 1967 by Delacrétaz *et al*., and mild superficial trauma and microangiopathy were thought to correlate with its development in genetically susceptible individuals.[1] Drug-induced acquired reactive

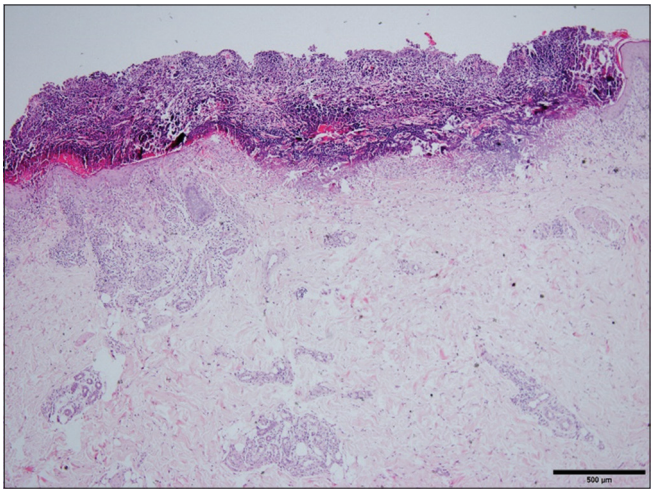

Figure 2a: A cup-shaped depression of the epidermis containing a keratin plug (H and E, ×400)

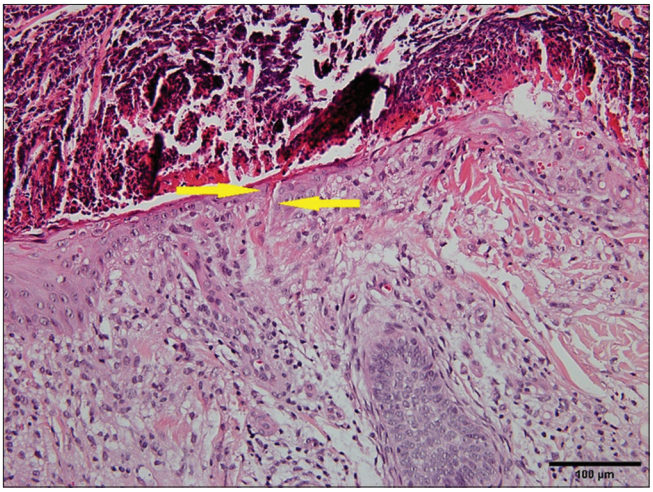

Figure 2b: Degenerated collagen fibers were eliminated through the epidermis (H and E, ×200) (arrows indicate the collagen fibers)

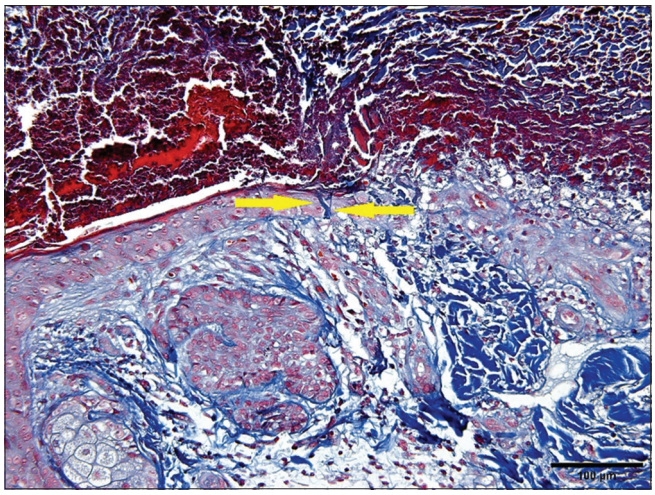

Figure 2c: Degenerated collagen fibers were eliminated through the epidermis (Masson trichrome, ×200) (arrows indicate the collagens)

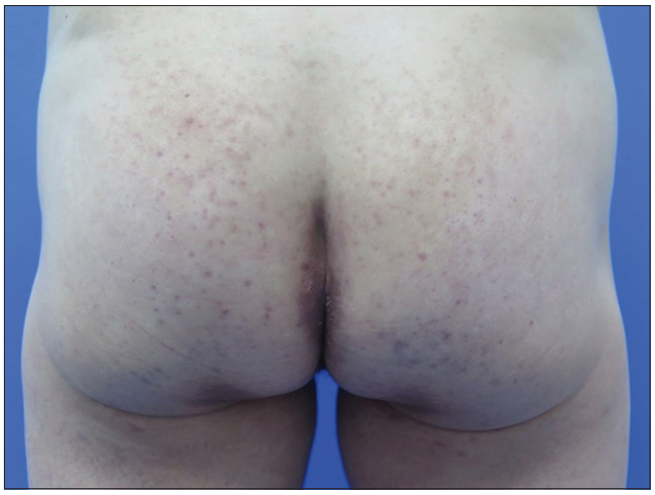

Figure 3b: Significant improvement in lesions on waist, buttocks and upper thigh after 2-month therapy with oral isotretinoin

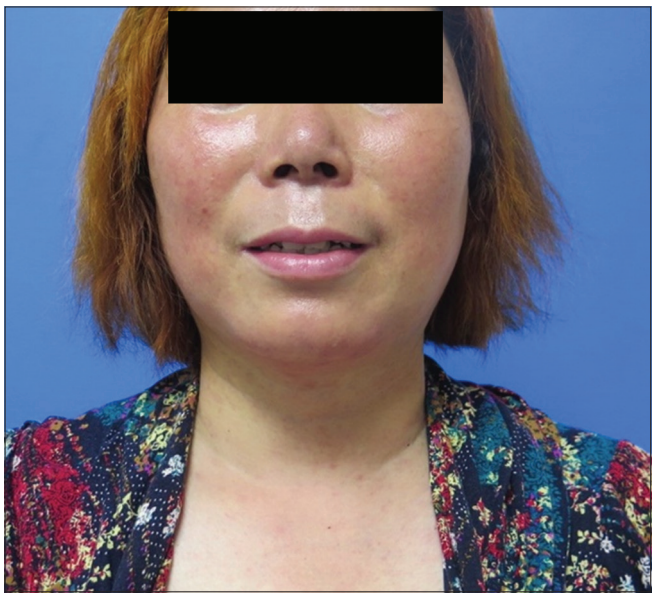

Figure 3a: Significant improvement in lesions on face and upper torso after 2-month therapy with oral isotretinoin

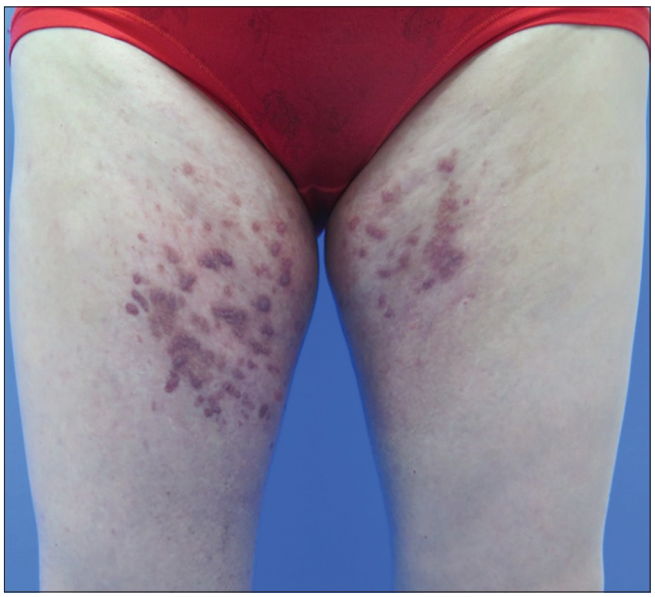

Figure 3c: Significant improvement in thigh lesions after 2-month therapy with oral isotretinoin

perforating collagenosis was occasionally reported with ranibizumab and sirolimus, but reasonable explanations were lacking.[2,3] Erlotinib, the epidermal growth factor-receptor tyrosine kinase inhibitor, acts in the treatment of non-small-cell lung cancer by inhibiting the proliferation, adhesion, migration and apoptosis of epidermal growth factor-receptor-expressed tumor cells. Epidermal growth factor-receptor is also wildly expressed in the basal layer of the epidermis, outer root sheath of the hair follicles, sebaceous and sweat gland apparatus. m-TOR, the downstream effector of epidermal growth factor-receptor signaling, plays a pivotal role in acne onset which might explain why acneiform eruptions occur the earliest and is common in the settings of erlotinib administration.[3-5] Given the actions of erlotinib, the inhibition of epidermal growth factor-receptor signaling might disturb the differentiation of keratinocytes, thus causing disruption of epidermis and follicular epithelium, contributing to the longer perforating process related to acquired reactive perforating collagenosis. Therefore, acneiform eruptions and acquired reactive perforating collagenosis may be taken together as two different stages of pathological changes in the skin induced by erlotinib, and a good response to systemic retinoids also corroborates the diagnosis, as in the present case. Acquired reactive perforating collagenosis following the occurrence of acneiform eruptions during erlotinib administration in lung adenocarcinoma is unusual and important for the dermatologists to recognize, in patients with epidermal growth factor-receptor inhibitor treatment.

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Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

Conflicts of interest

There are no conflicts of interest.

Xia Jiang, Ting-Ting Song, Fei Hao

Dermatology and Plastic Surgery Center, The Third Affiliated Hospital of Chongqing Medical University (General Hospital), Chongqing, China

Corresponding author:
Dr. Fei Hao,
No. 1, Shuanghu Branch Road, Yubei District, Chongqing 401120, China.
haofei@hospital.cqmu.edu.cn

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An unusual case of co-localization of proliferating trichilemmal tumor and seborrheic keratosis

Sir,

The incidence of benign and malignant lesions associated with seborrheic keratosis is about 9% and is proportionately linked with sun exposure.[1] Trichilemmal keratinization is the abrupt transition of nucleated epithelium of outer root sheath of the hair follicle to anucleated keratin without the formation of granular layer. Trichilemmal tumors arising in seborrheic keratosis are rare.[2-4] We report a case of proliferating trichilemmal tumor that developed in a long-standing seborrheic keratosis on the abdomen.

A 52-year-old woman presented with a progressively enlarging conical growth of five months duration arising from a raised black lesion on her lower abdomen that had been present for 20 years. Physical examination revealed a well-demarcated black colored verrucous plaque, measuring 5.7 cm × 3.5 cm in size, with a stuck-on appearance on the left iliac region . Arising from the edge of the plaque, was a hyperkeratotic scaly cutaneous horn-like growth of size 1.5 x 0.5 cm with an erythematous base [Figure 1]. There was no lymphadenopathy. Wide excision with 1 cm margin was performed, with the differential diagnosis of seborrheic keratosis for the black

Figure 1: Hyperkeratotic scaly cutaneous horn-like growth arising from a verrucous black colored plaque on the left iliac region

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